Spinal Cord Infarction Is an Unusual Complication of Intracranial Neuroendovascular Intervention

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Summary

Spinal cord infarction is an unusual complication of intracranial neuroendovascular intervention. The authors report on two cases involving spinal cord infarction after endovascular coil embolization for large basilar-tip aneurysms. Each aneurysm was sufficiently embolized by the stent/balloon combination-assisted technique or double catheter technique. However, postoperatively, patients presented neurological symptoms without cranial nerve manifestation. MRI revealed multiple infarctions at the cervical spinal cord. In both cases, larger-sized guiding catheters were used for an adjunctive technique. Therefore, guiding catheters had been wedged in the vertebral artery (VA). The wedge of the VA and flow restriction may have caused thromboemboli and/or hemodynamic insufficiency of the spinal branches from the VA (radiculomedullary artery), resulting in spinal cord infarction. Spinal cord infarction should be taken into consideration as a complication of endovascular intervention for lesions of the posterior circulation.

Introduction

Spinal cord infarction is an unusual complication of intracranial neuroendovascular intervention. The authors report on two cases involving spinal cord infarction in endovascular coil embolization for large basilar-tip aneurysms. In both cases, the wedge of the vertebral artery (VA) by the guiding catheter and flow restriction might have caused the thromboemboli and/or hemodynamic insufficiency of the spinal branches from the VA, resulting in spinal cord infarction.

Case Reports

Case 1

History and Examination

A 66-year-old man had a ten-year history of unruptured basilar-tip aneurysm that had shown gradual enlargement. It was round in shape and approximately 13 mm diameter with bilateral posterior cerebral arteries (PCA) branched at the aneurysm neck (Figure 1A). The bilateral VAs were almost equivalent in diameter, and the anterior spinal artery (ASA) originated from the left VA (Figure 1D). A left vertebral angiogram disclosed the C6 radiculomedullary artery branching from the left VA and filling the cervical segmental part of the ASA (Figure 1E, F).

Operation and Postoperative Course

Endovascular coil embolization was performed with an adjunctive stent/balloon combination-assisted technique [1]. Embolization was performed after local anesthesia and mild consciousness sedation. The left VA underwent catheterization using a 7-Fr guiding catheter

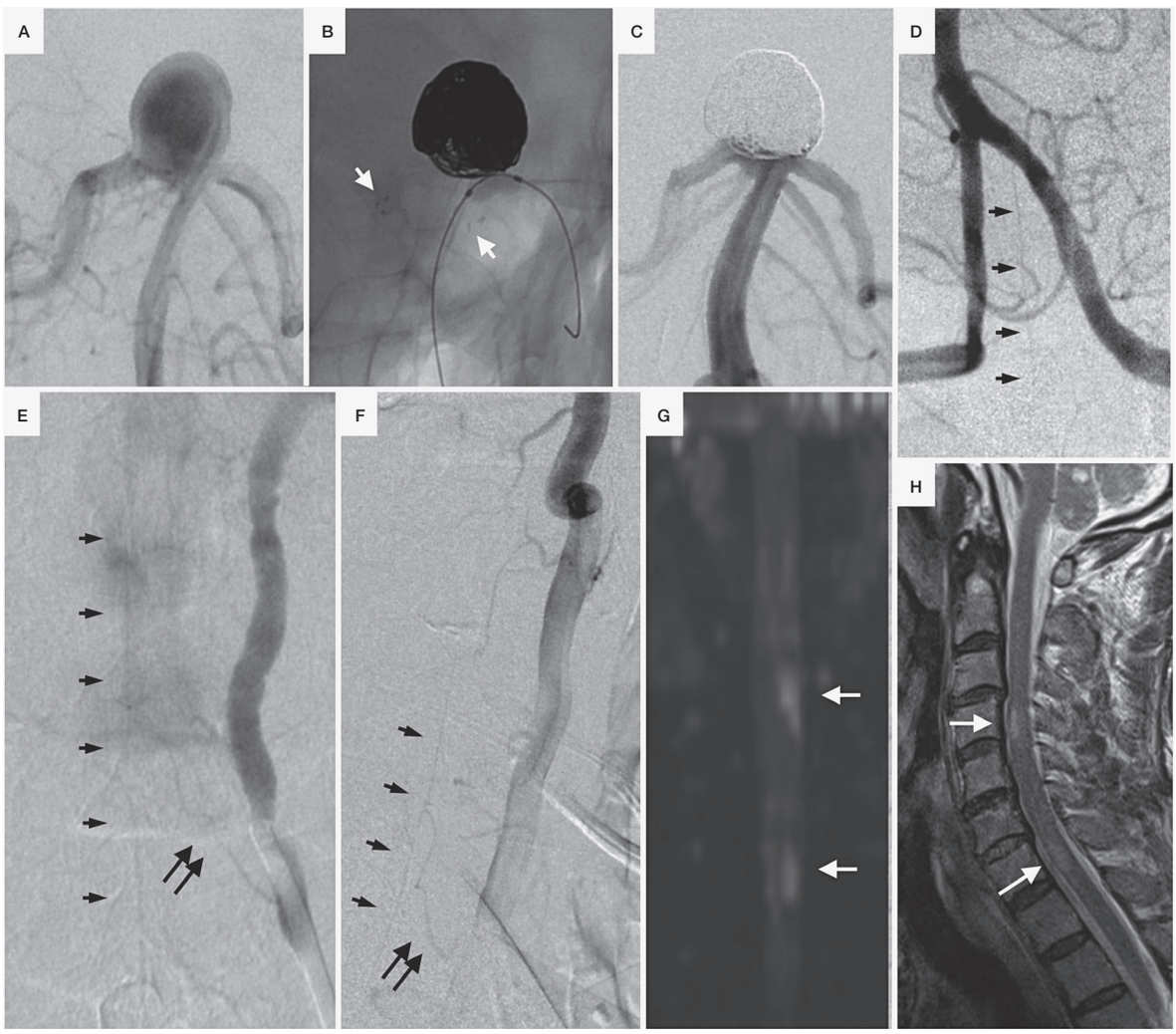
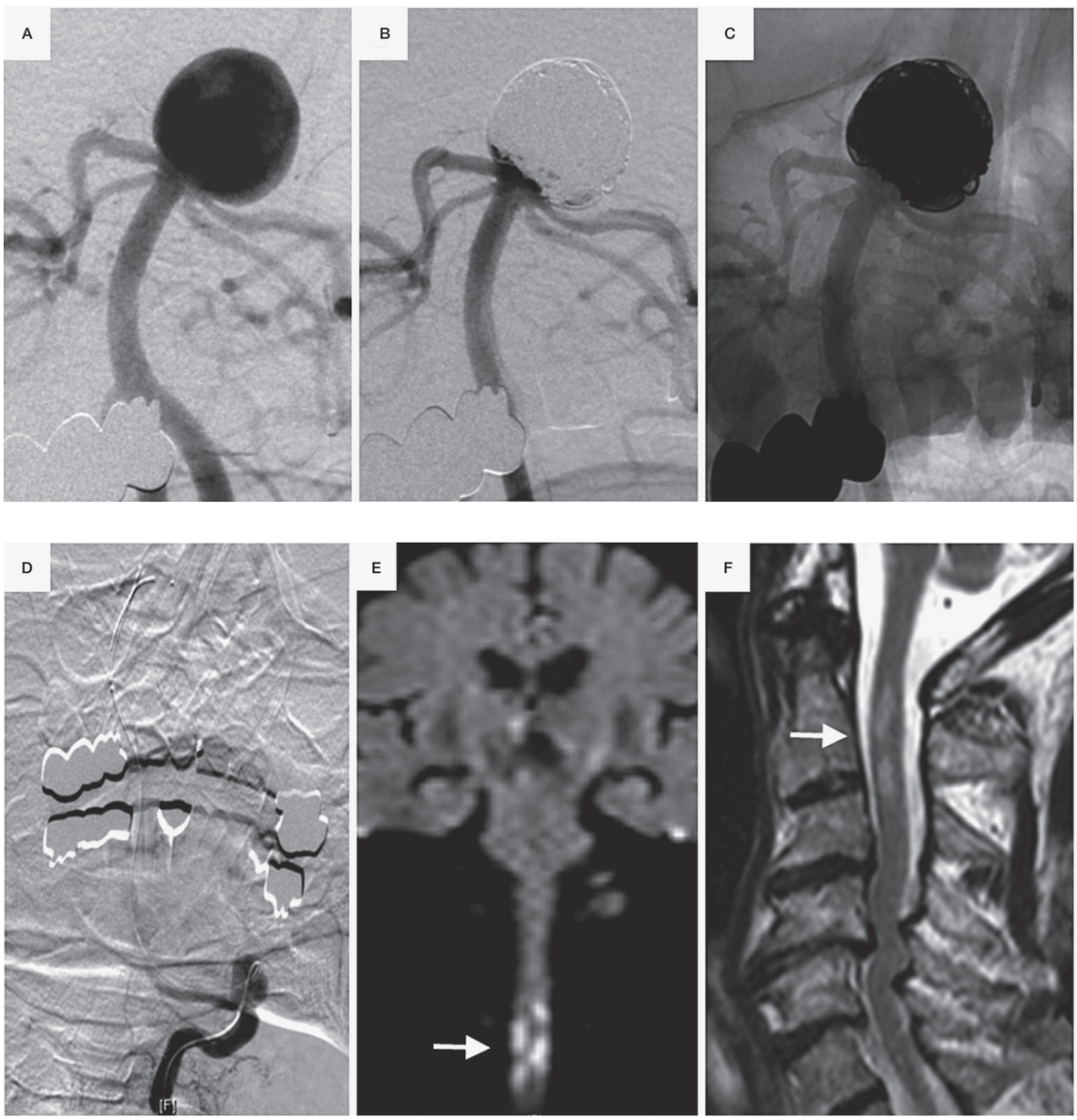


Figure 1 A wide-necked unruptured basilar-tip aneurysm was thoroughly embolized with a stent/balloon combination assist technique. A) Left vertebral angiogram (Lt. VAG) before embolization. B) Non-subtraction image during coiling showing balloon (arrow) and stent (arrows). C) Lt. VAG after embolization showing that the aneurysm was embolized and bilateral PCAs were successfully preserved. D) Anterior spinal artery (ASA) originating from left VA. E,F) Radiculomedullary artery (artery of cervical enlargement) (double arrow) is derived from the C6 level of the VA, and is filling the ASA (arrows), G) MRI diffusion weighted image acquired 1 day after the operation demonstrating multiple high intensity areas located mostly on the left side. H) MRI T2 weighted image showing spinal infarction at the C3 and C6 levels (arrow).

(Brite Tip, Cordis Endovascular, Miami, FL, USA) that was introduced from the right femoral artery. The authors selected the 7-Fr guiding catheter to advance simultaneously a stent-delivery catheter (Prowler Select Plus, Codman Neurovascular, Miami, FL, USA) and a balloon microcatheter (HyperForm 4 mm- 7 mm, ev3 Neurovascular, Irvine, CA, USA) for this adjunctive technique. The tip of the guiding catheter was wedged and catheter-induced vasospasm was observed just after navigating the guiding catheter into the VA (Figure 1E). Therefore, the catheter was slightly withdrawn to decrease the wedge. Vasodilators (such as Nimodipine or Nicardipine) were not used. The guiding catheter was continuously flushed with heparinized normal saline during microcatheter manipulation. After placement of the stent (Enterprise VRD 4.5 mm-28 mm, Codman Neurovascular), a microcatheter for coiling was introduced into the aneurysm by a trans-cell approach. Thereafter, the aneurysm was thoroughly embolized with stent/balloon assistance (Figure 1B). A total of 16 coils were inserted into the aneurysm cavity, and the bilateral PCAs were successfully preserved (Figure 1D).



Both the ASA and radiculomedullary artery were visualized after embolization. Dual antiplatelet therapy had been initiated one week before the operation. Heparin was administered with routine use during the procedure. Antiplatelet and anticoagulant therapy was continued postoperatively. It took approximately 3.5 hours to finish the operation.

Approximately five hours after the intervention, symptoms due to spinal cord infarction, including left hemiparesis and mild paresis of the right upper limb, presented and deteriorated. Cervical spine MRI diffusion and T2

Figure 2 A wide-necked ruptured basilar-tip aneurysm was totally embolized with a double catheter technique. A) Right vertebral angiogram (Lt. VAG) before embolization. B,C) Rt. VAG after embolization displaying the aneurysm was filled with coils and bilateral PCAs were successfully preserved (B: subtraction and C: non-subtraction images). D) Lt. VAG during procedure showing the flow stagnation of the left vertebral artery due to wedged guiding catheter and vasospasm. E) MRI diffusion weighted image acquired 2 days postoperatively demonstrating multiple high intensity areas bilaterally in the spinal cord. F) MRI T2 weighted image showing spinal cord infarction at the C2/3 level (arrow) and cervical canal stenosis at the C3-5 level.

weighted images revealed multiple high intensity areas in C4 and C7 portions (mostly in the left side), indicating the spinal cord infarctions (Figure 1G,H). Vessel dissection or any other lesion of the VA was not detected on the MR angiogram. The right upper limb paresis improved although left hemiparesis, sensory disturbance and incontinence persisted. The patient was transferred to a rehabilitation hospital after a two-week hospital stay.

Case 2

History and Examination

A 69-year-old man was transferred to the emergency department complaining of severe sudden onset of headaches with vomiting, and a CT scan revealed intraventricular and a small amount of subarachnoid hemorrhage. Three-dimensional CT angiogram showed a basilar-tip aneurysm. This aneurysm was also a large round aneurysm with a very wide-neck and its size was approximately 12 mm diameter (Figure 2A). The ASA originated from the left VA although it was faintly visualized. Radiculomedullary arteries from the cervical segment of the left VA were not clearly demonstrated angiographically in this patient. The lower cervical segment of the ASA was lightly filled by tiny radiculomedullary arteries branching from the right VA and cervical arteries (Figure 1E, F).

Operation and Postoperative Course

Endovascular coil embolization was performed with an adjunctive double-catheter technique to preserve the bilateral PCAs and to fill coils more tightly in the aneurysm. Embolization was performed after local anesthesia and deep consciousness sedation. A 7-Fr sheath was introduced from the right common femoral artery. The left VA underwent catheterization using a 7-Fr guiding catheter (Brite Tip), the same as Case 1. The authors selected the larger guiding catheter to decrease the interaction of two microcatheters for the adjunctive technique. The guiding catheters had been wedged in the VA during the procedure causing flow stagnation in the VA that was deemed to be acceptable (Figure 2D). Vasodilators were not used and the guiding catheter was continuously flushed with heparinized saline. Consequently, an additional diagnostic 4-Fr catheter was placed at the right VA for an intraoperative angiogram.

The aneurysm was sufficiently embolized by the double-catheter technique with two 18-type microcatheters (Excelsior 1018, Stryker Neurovascular, Fremont, CA, USA). A total of 50 coils were inserted into the aneurysm cavity, and the bilateral PCAs were preserved with the aneurysmal neck (Figure 2B,C). Heparin was administered with minimum use during the procedure, and intravenous-use antiplatelet agent was added at the final stage of the embolization. Antiplatelet therapy was continued postoperatively. It took approximately five hours to finish the operation. Postoperatively, consciousness sedation was continued overnight to manage the patient's condition.

On the day after the intervention, the patient presented left hemiparesis, and right hemiparesis followed the day after (tetraparesis). In contrast, he did not have any cranial nerve symptom. Brain MRI showed two small diffusion high spots, but no specific lesion intracranially. However, a cervical spine MRI revealed multiple infarctions at the spinal cord, bilaterally (Figure 2E,F). Cervical canal stenosis was observed in the C3-5 level. Anticoagulant and antiplatelet therapy was enforced after the diagnosis of spinal infarction. The right upper limb almost fully recovered and the left upper limb gradually improved. Paraparesis also improved but still remained. Finally, the patient changed hospitals to continue rehabilitation.

Discussion

Spinal cord infarction results from obstruction of the arteries that supply it, and is usually caused by arteriosclerosis, aortic disease, such as aortic aneurysm and dissection, or spinal vascular disease [2,3]. These involve the major arteries feeding the spinal cord to ischemia. Symptoms, which generally appear within minutes or a few hours of the infarction, may include paraparesis/tetraparesis, sensory disturbance, and incontinence [4]. Many reports have underscored spinal cord infarctions due to complications of open/endovascular aortic repair [5,6] or spinal surgery [7], and several have described VA dissection as a possible cause of spinal cord infarction [8]. However, there have been no reports of such complications caused by intracranial neurointervention. In our cases, the wedged guiding catheter in the VA may have induced cervical spinal cord infarctions due to thromboemboli and/or hemodynamic insufficiency of the radiculomedullary artery.

The arterial supply of the spinal cord can be provided from the ASA and the posterior spinal arteries (PSA). The pial arterial plexus connects ASA and PSA by surrounding the spinal cord. Although they have no precapillary connections, they have some overlapping anastomoses inside the spinal cord. The ASA is frequently duplicated for short distances in the cervical region, and central arteries that stem from a duplicated portion of the ASA supply only unilaterally [9-11]. Heterogeneity and laterality of the ischemic lesion in our cases might be caused by such anastomotic and anatomical variations. The radicular arteries supplying the cervical spine are delivered from intervertebral branches of the VAs and the cervical ASA receives several anterior radicular arteries (radiculomedullary arteries). Among them, the larger radiculomedullary artery is called the "artery of cervical enlargement" and usually arises from the lower cervical VA, but also from the deep or ascending cervical arteries [7,9,11]. Occlusion of an artery feeding the cervical regions rarely results in an infarction, as these areas have well-vascularized networks. However, the scarcity of anterior radicular arteries and the extensive distance between them might indicate that occlusion of a single radicular artery can result in spinal cord ischemia that is typically observed in the thoracic region (occlusion of the artery of Adamkiewicz). Similarly, insufficiency of the artery of cervical enlargement could result in a spinal cord infarction. Additionally, hemodynamic insufficiency of arteries feeding the spinal cord could be a risk for ischemia. In our cases, vascularized collaterals of the cervical spine may have been deficient.

Few papers have described lumbar spinal cord infarction after coronary angiography. Those reports speculated that inappropriate guidewire/catheter manipulation at the level of iliac artery might cause such a complication [12-14]. In our cases, improper handling of the guidewire/catheter was not found when advancing the guiding catheter except for the wedge of the catheter and catheter-induced vasospasm. In both cases, larger-sized guiding catheters were used for the adjunctive technique of coil embolization. Therefore, guiding catheters had been somewhat wedged in the VA during the procedure. Wedged VA with flow restriction may have caused the thromboemboli and/or hemodynamic insufficiency of the spinal branches from the VA. The authors speculate that thromboemboli of the "artery of cervical enlargement" mainly caused the spinal cord infarction in *Case* 1, and hemodynamic insufficiency might have affected arterial supply to the spinal cord in *Case* 2. Composite catheter-manipulation might affect the activation of thrombogenesis in both cases. Prolonged operation time and hypercoagulability after hemorrhage might also have contributed in *Case* 2, and cervical canal stenosis might have had some adverse effect on the vascular collaterals.

The authors are convinced that spinal cord infarction is a possible complication of endovascular intervention for lesions of the posterior circulation, and should be taken into consideration if neurological deficits without any cranial nerve manifestation are observed. It is recommended that the wedge of the VA should be avoided to prevent such a complication. The combination of proper-sized guiding catheters, placed in both VAs, substitutes for a larger-sized guiding catheter, appropriate use of vasodilators, shortening of the procedural time and adequate antiplatelet/anticoagulant therapy may contribute to preclude thrombogenesis in affected arteries.

Conclusion

Cervical spinal cord infarctions due to thromboemboli and/or hemodynamic insufficiency of the radiculomedullary artery induced by a wedged guiding catheter in the VA were demonstrated. Spinal cord infarction should be taken into consideration as a complication of endovascular intervention for lesions of posterior circulation.

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